

Dietary advice for the prevention of type 2 diabetes mellitus in adults (Review)

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[Intervention Review]

Dietary advice for the prevention of type 2 diabetes mellitus in adults

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Editorial group: Cochrane Metabolic and Endocrine Disorders Group.

Publication status and date: New, published in Issue 3, 2008.

Review content assessed as up-to-date: 31 October 2007.

Citation: Nield L, Summerbell CD, Hooper L, Whittaker V, Moore H. Dietary advice for the prevention of type 2 diabetes mellitus in adults. *Cochrane Database of Systematic Reviews* 2008, Issue 3. Art. No.: CD005102. DOI: 10.1002/14651858.CD005102.pub2.

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ABSTRACT

Background

Prevention of type 2 diabetes in adults is a far better option than treatment, to alleviate pressure on health care providers and resources. However, there is no current review of the evidence regarding the efficacy of a diet-only intervention for prevention.

Objectives

To assess the effects of type and frequency of dietary advice for the prevention of type 2 diabetes mellitus.

Search strategy

We carried out a comprehensive search of *The Cochrane Library*, MEDLINE, EMBASE, CINAHL, AMED, bibliographies and contacted relevant experts.

Selection criteria

All randomised controlled trials, of twelve months or longer, in which dietary advice for the prevention of type 2 diabetes was the only intervention in adults.

Data collection and analysis

The lead investigator performed all data extraction and quality scoring with duplication being carried out by one of the other four investigators independently with discrepancies resolved by discussion and consensus. Authors were contacted for missing data. Change data are presented.

Main results

Two trials which randomised 358 people to dietary treatment and control groups were identified. Longest duration of follow-up was six years.

In the 6-year Da Qing IGT & Diabetes study, the incidence of type 2 diabetes in the control group was 67.7% (95% confidence interval (CI) 59.8% to 75.2%) which was reduced to 43.8% (95% CI 35.5% to 54.7%) in the diet group. Overall, the dietary intervention group had a 33% reduction in the incidence of diabetes after six years ($P < 0.03$). The Oslo Diet & Exercise Study (ODES) found

significant ($P < 0.05$) reductions in insulin resistance, fasting insulin (pmol/L), fasting C-peptide (pmol/L), fasting proinsulin (pmol/L), fasting blood glucose (mmol/L), BMI (kg/m^2), mBP (mmHg) and fasting triglycerides (mmol/L), and a significant increase in fasting HDL cholesterol (mmol/L) and PAI-1 (U/ml) after 12 months of dietary intervention.

Data on mortality, morbidity, health-related quality of life, adverse effects, costs were not reported in either study.

Authors' conclusions

There are no high quality data on the efficacy of dietary intervention for the prevention of type 2 diabetes. More well-designed, long-term studies, providing well-reported, high-quality data are required before proper conclusions can be made into the best dietary advice for the prevention of diabetes mellitus in adults.

PLAIN LANGUAGE SUMMARY

Dietary advice for the prevention of type 2 diabetes mellitus in adults

Two trials randomised 358 participants to dietary advice and control treatment groups. The longest duration of dietary advice was six years, the only other trial lasted 12 months. Dietary advice appears to be effective in reducing the risk of diabetes by 33% compared to control group over six years. After 12 months, dietary advice appears to beneficially effect indicators of metabolic control. Data on mortality, morbidity, health-related quality of life, adverse effects and costs were not reported.

BACKGROUND

Description of the condition

Diabetes mellitus is a metabolic disorder resulting from a defect in insulin secretion, insulin action, or both. A consequence of this is chronic hyperglycaemia (elevated levels of plasma glucose) with disturbances of carbohydrate, fat and protein metabolism. Long-term complications of diabetes mellitus include retinopathy, nephropathy and neuropathy. The risk of cardiovascular disease is increased. For a detailed overview of diabetes mellitus, please see under 'Additional information' in the information on the Metabolic and Endocrine Disorders Group on the Cochrane Library (see 'About the Cochrane Collaboration', 'Collaborative Review Groups-CRGs'). For an explanation of methodological terms, see the main Glossary in *The Cochrane Library*.

Type 2 diabetes mellitus is a very common chronic disease. The prevalence of the disease is increasing across the world and is linked with the adoption of a 'Western' lifestyle, mainly in terms of dietary habits and physical activity (Torjesen 1997). Around 80% of individuals who develop type 2 diabetes are initially obese (Hensrud 2001), which is known to further contribute to an increased insulin resistance (Pi-Sunyer 2000). There is a strong familial predisposition to developing the disease; first-degree rela-

tives of known type 2 diabetic patients are at an increased risk of developing the disorder (Martin 1992), indicating that in some cases, people are genetically predisposed to this disease. However, little is known about the genetics of diabetes and some studies have shown that environmental and lifestyle factors are much more important determinants. Therefore, a wide-scale genetic screening strategy would be ineffective and wasteful to resources. Ageing and increased sedentary behaviour are also associated with the appearance of the disease. A new cause for concern is that recently type 2 diabetes has started to affect the obese paediatric population (Sinha 2002).

There are an estimated 2.35 million people with diabetes in England, with up to another 750,000 undiagnosed cases and this is predicted to grow to more than 2.5 million by 2010 (DOH Website; Diabetes UK Website). Type 2 diabetes mellitus is the more common of the two main types and accounts for between 85% to 95% of all diabetic patients (Diabetes UK Website). In the United Kingdom alone, treatment of type 2 diabetes costs around £1.8 billion per annum (Moore 2000), which is predicted to grow if this epidemic is left alone without implementation of a strong, effective and strategic prevention program. In the World Health Organisation report (WHO/FAO 2003), it was suggested that the changes needed to reduce the risk of developing type 2 diabetes

at a population level would be unattainable unless substantial environmental changes to facilitate individual choices were implemented.

Risk factors for type 2 diabetes include population group, age, obesity, waist-to-hip ratio, low birth weight, family history of diabetes, history of gestational diabetes and physical inactivity. However, the precise detail of how these risk factors affect the pathophysiology of disease is not well understood (Goldberg 1998). It is estimated that newly diagnosed diabetic patients have been hyperglycaemic for 5 to 10 years before diagnosis, with 10% to 20% already having some tissue complications (Goldberg 1998). Improved understanding of the development of type 2 diabetes suggests that this disease has a prolonged prediabetic phase, giving hope that individuals who are at high risk of becoming diabetic might be identified before the disease progresses and given preventative treatments.

Insulin resistance is usually the first clinically detectable stage in the evolution of type 2 diabetes (Goldberg 1998) and in theory, screening on this basis would appear particularly interesting as this may lead to practices that could reduce insulin resistance and therefore retard or prevent the development of type 2 diabetes and its associated complications. However, it is estimated that although 25% of the population would be defined as insulin resistant, only 5% to 6% of the population would develop diabetes (Goldberg 1998). All individuals who develop type 2 diabetes pass through the impaired glucose tolerance (IGT) phase, caused by malfunctioning β -cells, and defined by the World Health Organisation (WHO) as a fasting glucose $<140\text{mg/dL}$ and a 2-hour glucose value of 140 to 199mg/dL after 75g of oral glucose. Factors which increase the risk of progression from IGT to type 2 diabetes include high fasting and 2-hour glucose measurements, glycated haemoglobin, ethnicity and obesity, but the most important determinant is the severity of the glucose intolerance (Goldberg 1998).

Description of the intervention

A person with newly diagnosed diabetes is usually offered time with a dietitian who will show them how to reduce their fat intake and increase their complex carbohydrate intake, although dietary advice can be offered by any trained health care professional. However, by this stage the disease has already been diagnosed and it is too late for any prevention strategies. The dietitian can only treat the disease and help to prevent, or slow down, the progression of diabetes towards more serious complications. An effective prevention mechanism is required to suppress this epidemic as 25% to 75% of those with IGT, develop diabetes within a decade of their diagnosis (Saad 1988)

Diets commonly used to control blood glucose levels include low fat and high unrefined carbohydrate, (those which take around 25% to 30% of energy from fat and around 50% of the total energy from unrefined carbohydrate), or low glycaemic index (GI) diets (foods that have a low glycaemic index include pasta products,

oats, beans and some fruits and vegetables) usually both in combination with weight reducing advice. Diets based on glycaemic index of food have lately come into vogue, and Brynes published a letter reporting a short trial which showed that a low glycaemic index diet significantly improved the 24-hour blood glucose profile in type 2 diabetics (Brynes 2003). Two systematic reviews in this area for the Cochrane Collaboration published conflicting recommendations, regarding the usefulness of low GI diets (Kelly 2004; Thomas 2007). More recent research has also looked into the effect of individual macro- and micro-nutrients such as fibre, chromium, and so called 'functional foods', but much of this evidence seems to be incomplete, with relatively few intervention trials carried out long-term (Riccardi 2005). However, it is thought that similar diets may be of value in those with metabolic syndrome in preventing or delaying progression to frank diabetes (Riccardi 2000). The use of a high polyunsaturated or high monounsaturated fat diet has been investigated with regards to weight and diabetes. In 1998, Garg reviewed the use of high monounsaturated fat diets compared with traditional high-carbohydrate diets and found that the monounsaturated fat diet actually both increased HDL and reduced fasting triglycerides and VLDL-cholesterol (Garg 1998). Brynes et al. published a report of a short trial, which did not show any association between an increase in dietary fat and a change in insulin sensitivity (Brynes 2000).

Why it is important to do this review

There is now unequivocal evidence that type 2 diabetes can be prevented or at least delayed by interventions including a dietary component. Risk of such progression was decreased by over half in the two major trials to date (DPP 2002b; Tuomilehto 2001). The United States Diabetes Prevention Program, (which was ineligible for inclusion in this review), reported a reduction of 58% (95% confidence interval (CI) 48% to 66%) in the incidence of diabetes when participants were treated with the lifestyle intervention compared with a 31% reduction (95% CI 17% to 43%) of incidence of diabetes for the metformin-treated participants (DPP 2002b), which demonstrates that progression from impaired glucose tolerance to type 2 diabetes mellitus is not inevitable with appropriate lifestyle intervention.

However, we are not aware of the existence of any systematic reviews addressing the impact of dietary advice alone on the prevention of type 2 diabetes. A review by Anderson and colleagues (Anderson 2003), carried out on the subject of 'Importance of Weight Management in Type 2 Diabetes', suggested that "the United States health care providers should endorse the American Heart Association's and European Diabetes Association's recommendations that diabetic people .. achieve and maintain a BMI equal to or less than 25 kg/m^2 " and that "weight management may be the most important therapeutic task for most obese type 2 diabetic individuals".

Despite the large body of evidence supporting dietary treatments, the authors of this review also recognise the importance of lifestyle changes and the impact of physical activity. In general, support is given for a multifaceted approach to diabetes prevention. It is therefore advised that in practice, this review is read alongside other Cochrane diabetes prevention reviews such as 'Long-term non-pharmacological weight loss interventions for adults with prediabetes' (Norris 2007) and the upcoming review by Orozco et al which is in protocol format at the moment 'Exercise or exercise and diet for preventing type 2 diabetes mellitus' (Orozco 2007) which covers topics outside the scope of this review. However, given the prevalence of type 2 diabetes and the potentially serious costs (health status and financially) of the disease, it is important to establish which type of dietary advice, if any, is most effective in preventing development.

This Cochrane review therefore aims to assess the evidence that exists to establish what kind of dietary advice is effective in preventing development of type 2 diabetes mellitus in adults.

OBJECTIVES

To assess the effects of type and frequency of dietary advice for the prevention of type 2 diabetes mellitus.

METHODS

Criteria for considering studies for this review

Types of studies

All randomised controlled clinical trials (RCTs) that followed patients for at least twelve months were included. Randomisation of individuals or clusters of individuals were accepted.

Types of participants

All studies had to include adult participants who were 18 years or older.

To be consistent with changes that have occurred over time in the classification and diagnostic criteria of impaired glucose tolerance, the diagnosis should have been established using the standard criteria valid at the time of the beginning of the trial. Ideally, the diagnostic criterion was described in the article. Diagnostic criteria used included those described by the National Diabetes Data Group standards (NDDG 1979), the World Health Organisation standards (WHO 1980; WHO 1985; WHO 1998) and

the American Diabetes Association Standards (ADA 1997; ADA 1999; ADA 2002).

Studies performed on participants suffering from impaired glucose tolerance were included in this review. Where a study reported combined results for participants with type 2 diabetes and participants with impaired glucose tolerance, then efforts were made to contact the authors of the study to obtain individual patient data. Where this was unsuccessful, the trial was excluded.

Participants could be of either sex, but those who were acutely ill or pregnant were excluded.

Types of interventions

Studies where the intervention was dietary advice with an aim of reducing weight and risk of developing type 2 diabetes mellitus were included in this review. Studies that compared the effects of dietary advice versus no dietary advice or dietary advice versus different dietary advice were also included. Studies were not included if they included medication that was provided differently in the control and intervention groups.

Types of outcome measures

Primary outcomes

- Incidence of type 2 diabetes. Ideally, the diagnostic criteria for type 2 diabetes mellitus should have been described in the trial. To be consistent with changes in classification and diagnostic criteria of the disease through the years, the diagnosis should have been established using the standard criteria valid at the time of the beginning of the trial. Changes in the diagnostic criteria may have produced significant variability in the clinical characteristics of the patients included as well as in the results obtained. These differences were considered and planned to be explored in a sensitivity analysis;
- Glycaemic control measures (oral glucose tolerance test, both fasting and 2-hour and glycated haemoglobin; for the detection of impaired glucose tolerance);
- Time to development or diagnosis of type 2 diabetes mellitus.

Secondary outcomes

- Quality of life (ideally, measured using a validated instrument);
- Mortality;
- Morbidity;
- Weight;
- body mass index (BMI);
- Cost of intervention(s);

- Serum cholesterol (LDL and/or HDL) and serum triglycerides;
- Blood pressure;
- Maximal exercise capacity;
- Adverse effects.

Timing of outcome measurement

Randomised controlled trials of interventions that involved participants for a minimum of twelve months were included in the review. Outcome measures were extracted and assessed at baseline, one year, two year and at one year intervals from that point where available.

Search methods for identification of studies

Electronic searches

We used the following sources for the identification of trials: *The Cochrane Library*, which includes the Cochrane Central Register of Controlled Trials, the Database of Systematic Reviews and the Database of Abstracts and Reviews of Effectiveness (issue 4, 2007), MEDLINE (until November week 2, 2007), EMBASE (until week 50, 2007), CINAHL (until December week 1, 2007) and AMED (until December 2007). There were no language restrictions for either searching or trial inclusion.

The described search strategy (see for a detailed search strategy [Appendix 1](#)) was used for MEDLINE. For use with EMBASE, *The Cochrane Library* and the other databases this strategy was slightly adapted.

We also searched databases of ongoing trials: Current Controlled Trials (<http://www.controlled-trials.com> - with links to other databases of ongoing trials), UK National Research Register (<http://www.update-software.com/National/>); National Institutes of Health (<http://clinicalstudies.info.nih.gov/>).

Searching other resources

References of the 132 full text journal articles were screened for potentially relevant articles.

Data collection and analysis

Two reviewers undertook assessment of results data independently. Results data were assessed by the lead reviewer (LN) and duplicated by one of the co-reviewers (either HM, VW, CS or LH). Information on a number of measures of methodological quality of the included studies was assessed independently by two reviewers (LN plus one other from HM, VW, CS or LH); study design, method of allocation concealment, blinding of outcome

assessment and drop out rates. Where there was uncertainty, authors were contacted to clarify aspects of study design. Differences between reviewers' extraction results were resolved by discussion. Multiple publications were collated and assessed as one study.

Selection of studies

In the first instance, relevant studies were determined from the initial search of electronic databases, and then through screening by the lead reviewer (LN) and duplicated by one of the co-reviewers (either HM, VW, CS or LH). Articles were rejected during this initial screening if the reviewer could determine from the title or abstract that it did not meet the inclusion criteria, if rejection was not possible, full text copies were retrieved.

Data extraction and management

Two reviewers (the lead reviewer (LN) plus one other from either HM, VW, CS or LH) independently extracted data from each study using a data extraction form based on the one provided by the Metabolic and Endocrine Disorders Review Group. Differences between reviewers' extraction results were resolved by discussion, and where necessary, in consultation with a third reviewer. Data concerning participants, interventions, and outcomes, as described in the selection criteria section, were extracted. Trial quality characteristics, including method of randomisation, allocation concealment, blinding of outcome assessors and losses to follow-up, were extracted onto the form. In addition, data were collected on potential effect modifiers including age, presence of diabetic micro or macrovascular disease, blood pressure, lipids, body mass index and bodyweight.

Assessment of risk of bias in included studies

The quality of each trial was assessed based largely on the quality criteria specified by Jadad and Schulz ([Jadad 1996](#); [Schulz 1995](#)) by the lead reviewer (LN) and duplicated by one of the co-reviewers (either CS, HM, VW or LH). Interrater agreement was reviewed. In particular the following factors were looked at:

- (1) Minimisation of selection bias - score: 0 or 1 or 2.
 - (a) one point was given if the study could be described as randomised (which includes the use of words such as 'random', 'randomly' and 'randomisation');
 - (b) one additional point was given if the study described the method of randomisation and was an appropriate method;
 - (c) one point was taken away if the study described the method of randomisation, but was an inappropriate method.
- (2) Minimisation of detection bias - score: 0 or 1 or 2.
 - (a) one point was given if the study was described as blinded (in any capacity);
 - (b) one additional point was given if the method of blinding was described and was appropriate;

(c) one point was taken away if the method was described as blinding but was inappropriate.

(3) Minimisation of attrition bias - score: 0 or 1.

(a) one point was given if the withdrawals and dropouts from the study are described.

We did not score on performance bias.

Based on these criteria, studies were broadly subdivided into the following three categories (see Cochrane Handbook for Systematic Reviews of Interventions):

A - all quality criteria met: low risk of bias (studies with scores of four and five points (and with at least one point allocated from each section) were allocated to this category).

B - one or more of the quality criteria only partly met: moderate risk of bias (studies with scores of three points (and with at least one point in each section) were allocated to this category).

C - one or more criteria not met: high risk of bias (S' studies with scores of zero, one, two and three points were allocated to this category (that is studies with no points in at least one section)).

These subdivisions of the studies were used as the basis of a sensitivity analysis, if the included studies receive notably different quality scores. Quality scores and categories were reported in the 'characteristics of included studies' table.

Measures of treatment effect

Endpoint versus change data: Where possible, change data are presented. Change data were calculated from endpoint data - baseline data if only endpoint data were presented.

Data for the Da Qing study (Pan 1997) were presented with baseline and endpoint data (after six years), therefore the mean change data were calculated manually by the lead reviewer (checked by a co-reviewer) using endpoint - baseline = change (LN, HM), with baseline standard deviations assumed equal to endpoint.

There were no continuous outcomes, however, in the case of such data, a weighted mean difference (WMD) between groups would have been estimated.

Assessment of heterogeneity

A test for heterogeneity was planned, using the standard χ^2 -test, as well as by visually inspecting the graphs (the I^2 -test was planned to be used to quantify heterogeneity (Higgins 2002)). If there was little heterogeneity between trial results, data would be summarised statistically using a fixed effect model for continuous data. A significance level less than 0.10 would have been interpreted as evidence of heterogeneity. If heterogeneity was found, the data would be re-analysed using a random effects model to see if this made a substantial difference. However, because of the lack of data, there were no meta-analyses carried out, so testing for heterogeneity could not be carried out.

Subgroup analysis and investigation of heterogeneity

Subgroup analyses were planned a priori but were not undertaken as they were not applicable to the included studies. These subgroup analyses were planned to assess whether particular groups of people with type 2 diabetes could obtain more benefit from a particular intervention than other groups could. Efficacy of different combinations of types of diets would have also been considered in the subgroup analyses.

The main analysis carried out were as follows:

- Dietary advice versus a different dietary advice

Subgroup analyses for this comparison would have been:

- dietary advice in lean participants versus the same dietary advice in obese participants;
- with or without weight loss advice (outcome measures: development of (further) microvascular disease only);
- presence/absence of microvascular disease (outcome measures: development of (further) microvascular disease only).

However, due to there only being two papers included, there was insufficient data to carry out any subgroup analyses.

Sensitivity analysis

No sensitivity analyses were performed.

RESULTS

Description of studies

See: [Characteristics of included studies](#); [Characteristics of excluded studies](#).

Results of the search

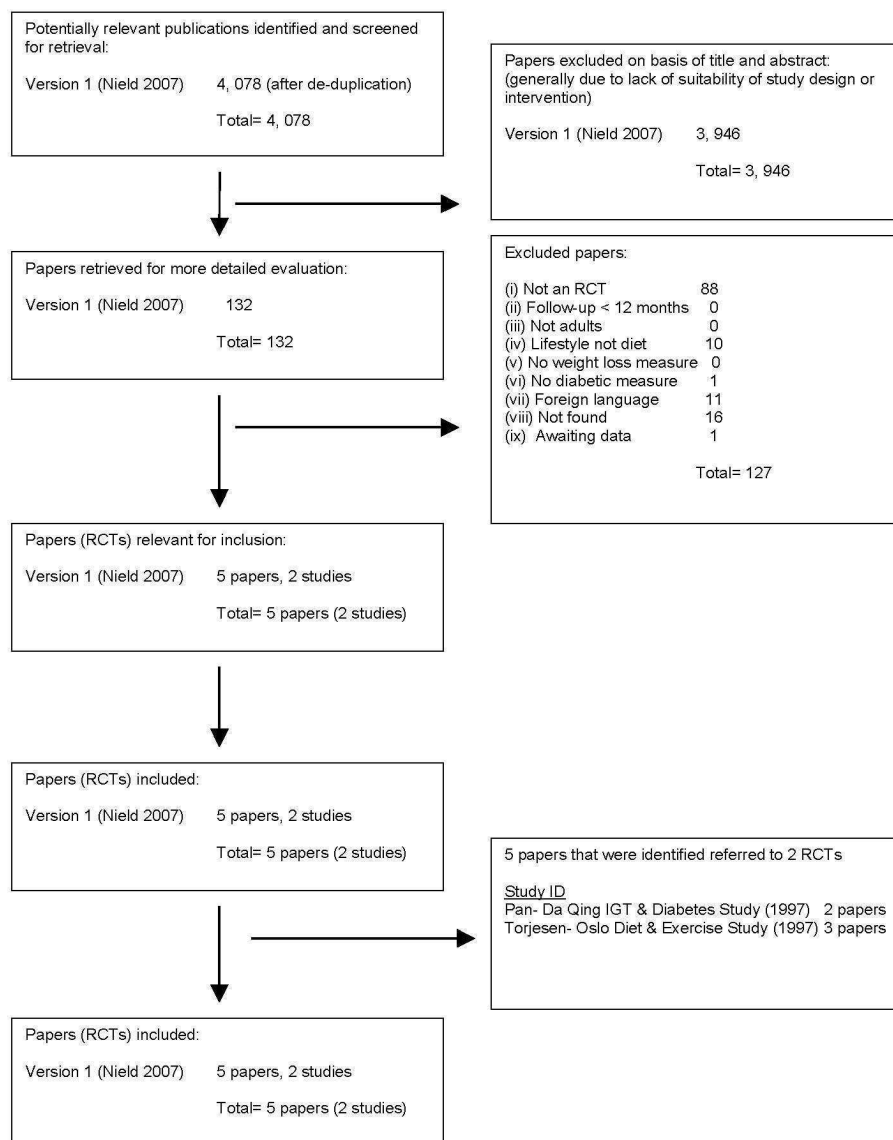
The search identified 4078 records, from these, 132 full papers were identified for further examination and assessed independently by two reviewers (LN plus one other from HM, CS, LH or VW). The other studies were excluded on the basis of their abstracts because they were not relevant to the question under study (see [Figure 1](#) for details of the amended QUOROM (quality of reporting of meta-analyses) statement). After screening the full text of the selected papers, five papers were found which met the inclusion criteria, referring to two studies; Da Qing IGT and Diabetes Study (Pan 1997) and the Oslo Diet and Exercise Study (ODES) (Torjesen 1997).

Figure 1.

GRAPHS AND OTHER TABLES

Figure 01: Quorum statement flow diagram – dietary interventions for preventing diabetes

4373 papers were found in the original search before duplication [14.12.07]



Assessment of publication bias inter-rater agreement

Inter-rater agreement for study selection, qualifying a study as 'included' or 'potentially' relevant, was very high between the lead reviewer (LN) and all co-reviewers (HM, CS, VW and LH).

Missing data

Attempts were made by the lead reviewer to contact the lead authors in the event of data which has been reported or recorded but not published in the text of the articles. A reply was received from Torjesen (Torjesen 1997). However, further information for the Pan (Pan 1997) study was untraceable. There was a third study (Swinburn 2001) which had potential to be included, but some participants were diabetic at the start of the study, attempts were made to contact the authors, but no further information could be retrieved.

Included studies

Two studies met the inclusion criteria. The Oslo Diet & Exercise Study (ODES) looked at an increased intake of fish and reduced total fat intake (Torjesen 1997), and the Da Qing IGT and Diabetes Study looked at prescription of a diet based upon body weight (25 to 30 kcal/kg), increased consumption of vegetables, and a reduction of simple sugars and alcohol (Pan 1997).

Interventions

The participants in the trial lead by Pan (Pan 1997) were randomised by clinic to one of four groups: diet only, diet plus exercise, exercise only and control. For the purpose of this review, only the diet and control groups were eligible for comparison. Participants with a body mass index (BMI) less than 25kg/m² were prescribed a diet containing 25 to 30kcal/kg body weight which consisted of 55% to 65% carbohydrate, 10% to 15% protein and 25% to 30% fat. These participants were asked to consume more vegetables, limit their alcohol consumption, and reduce their intake of simple sugars. Individuals with a BMI greater than 25kg/m² were encouraged to reduce their calorie intake in order to promote weight loss of 0.5 to 1.0 kg per month until they reached a target BMI of 23 kg/m². Targets were set for individuals for total calorie consumption, and quantity of cereals, vegetables, milk, meat and oils. This was promoted by the provision of a list of the recommended daily intake of commonly used foods and a substitution list which allowed exchanges within a food group. All participants received individual counselling by physicians concerning their daily food intake, with small group counselling sessions running weekly during the first month, monthly for the first three months, and three-monthly for the remainder of the study. Participants from clinics assigned to the control group were provided with general information about diabetes and impaired glucose tolerance (IGT). Physicians also handed out some leaflets with general instructions for diet and/or increased leisure time physical activity. No individual instruction or group counselling sessions were held.

In the Oslo Diet and Exercise Study (ODES) (Torjesen 1997), participants were again randomised to four groups: diet only, diet plus exercise, exercise only and control. The diet intervention was individualised and provided individually for each participant with their spouse (Holme 1993). The dietary counselling encouraged a decrease in the total caloric intake, included increased intake of fish and fish products and a reduced total and saturated fat intake. In addition, participants were advised to increase their intake of vegetables and fibre-rich complex carbohydrate products and a decrease their intake of sugar. Participants with high blood pressure were also requested to decrease their intake of salt. During the counselling sessions, a target body weight reduction was agreed on, typically of 0.5 to 1.0kg per month, but as high as 2 kg/month for the most overweight individuals. At the end of the session, each participant was given an individualised dietary programme, including the 5 to 10 most important points. Participants were asked to record their body weight weekly. Their dietary habits were followed up at three and nine months into the study. All participants in the control group, were asked not to change their dietary habits or lifestyle during the trial, but, as with all participants, they were asked to stop smoking.

Number of study centres

In the Da Qing IGT and Diabetes Study, there were 33 clinics involved (Pan 1997) compared with only one in the Oslo Diet and Exercise Study (Torjesen 1997).

Country and location

Of the two studies which compared dietary advice to a control group, one was based in Da Qing, Hei Long Jiang Province, China (Pan 1997) and one in Oslo, Norway (Torjesen 1997).

Setting

All of the participants were home-based or free-living but received dietary advice by attending a clinic (Pan 1997) or at a university hospital (Torjesen 1997).

Treatment before study

Participants for the Oslo Diet and Exercise Study (Torjesen 1997) were recruited from an ongoing screening program of 40-year old men and women in Oslo, participants in the Da Qing IGT and Diabetes Study (Pan 1997) agreed to take part in the randomised controlled trial after they were identified during an on-going population-based survey of diabetes and IGT in Da Qing, China.

Methods

Pan (Pan 1997) randomised participants by clinic for the controlled clinical trial to investigate the effects of diet, exercise and diet plus exercise on the incidence of diabetes in people with impaired glucose tolerance (IGT). Participants were also split into two sub-groups according to their BMI- 'overweight' was defined as participants with BMI greater than 25 kg/m² and 'lean' as participants with BMI less than 25 kg/m². After their initial screen-

ing, participants attended a general health check at three-month intervals with a local physician and discussed compliance with the intervention with nurses and clinical staff. At each three month follow-up visit, weight, blood pressure and urinary glucose were measured. If the urinary glucose appeared positive, further diagnostic tests were carried out. Firstly, plasma glucose was measured two hours after a standard breakfast of 100g steamed bread. If this postprandial glucose concentration was $>200\text{mg/dl}$ (11.1mmol/l), or the physician suspected that the participant had developed diabetes, the subject received a 75g oral glucose tolerance test at the hospital. If at any time during the study, a participant had symptoms of diabetes and repeated fasting plasma glucose measurements greater than 140 mg/dl (7.8 mmol/L) or a one-off glucose measurement was greater than 200 mg/dl (11.1 mmol/L), a clinical diagnosis of diabetes was made. If the participant met WHO criteria for diabetes following these test, their formal participation in the study ended. Every two years, all participants (including those diagnosed with diabetes at their 3-month check ups) underwent a systematic evaluation examination where their height, weight, blood pressure, fasting plasma glucose and 2-hour plasma glucose were reviewed. If results indicated diabetes (fasting glucose greater than 140 mg/dl [7.8 mmol/L] or 2-hour glucose greater than 200mg/dl [11.1 mmol/L]) then the oral glucose tolerance test was repeated 7 to 14 days later. If diagnosis of diabetes was confirmed, the participant was referred to standard diabetes treatment. If the repeat results were below diabetic levels, then the participant continued in the study.

The Oslo Diet and Exercise Study (Torjesen 1997) was a 2x2 randomised primary preventative trial, forming four groups- diet, exercise, diet and exercise and control groups from the two interventions- physical exercise and dietary change. All participants were selected from a database of men and women who complied with a number of inclusion criteria. Insulin, C-peptide, proinsulin and glucose were measured at baseline and at 12 months from serum drawn before and 60-minutes after a 75g standard oral glucose load. All measurements were determined from one assay series for each participant. Analyses were performed in batches at the end of the trial from frozen samples. Insulin was assayed by a radioimmunoassay using an antibody which had no cross-reaction against pro-insulin or des 31,32 split pro-insulin. C-peptide was assayed by radioimmunoassay with a 5% cross-reaction against pro-insulin (provided by Diagnostic System Laboratories, Webster, Texas). Pro-insulin was measured by an immunometric assay using monoclonal antibodies against the insulin and C-peptide parts. Other components were measured using standard methods. Insulin resistance and relative β -cell function were calculated from the fasting serum levels of glucose and insulin. Insulin resistance and relative β -cell for each participant was determined using formulae. Each participant was examined for all risk factors at baseline and year 1 of the study. This included an assessment of family history, patient history of earlier disease, a description of exercise, eating and smoking habits and a full physical examination. Some

measurements such as cholesterol, HDL-cholesterol and triglycerides were also taken at three and nine months for those who were in the dietary advice group.

Duration of the intervention

Recruitment for the Pan et al. study (Pan 1997) was completed in 1986 and the intervention was carried out for six years. Participants received individual counselling weekly for the first month, monthly for the first three months, and three-monthly for the rest of the study. Systematic evaluation assessments were carried out every two years in 1988, 1990 and 1992.

Participants were involved in the intervention period for a duration of 12 months in the Oslo Diet and Exercise Study (Torjesen 1997).

Duration of follow-up

Follow-up of the Pan study (Pan 1997) was continued for six years, at two year intervals in 1988, 1990 and 1992, with data published for baseline and 6-year endpoint.

In the Oslo Diet and Exercise Study, follow-up was only continued for one year with data collected at baseline and one-year time points (Torjesen 1997).

Run-in period

There were no run-in periods reported for either of the two studies.

Language of publication

Both of the studies were published in English (Torjesen 1997 and Pan 1997).

Participants

The two trials followed 358 participants. There were 176 in the control groups and 182 in the intervention groups. The samples used in these trials were generally representative of the population, as the majority were recruited from ongoing mass screening programmes.

Who participated

Participants in the Oslo Diet and Exercise Study (Torjesen 1997) were recruited from an on-going screening programme, all participants were aged 41 to 50 years old and were male and female. Male ($n = 132$) and female ($n = 131$) participants over the age of 25 were recruited for the Da Qing IGT and Diabetes Study (Pan 1997) for the diet and the control groups. For the control group, mean age was 46.5 ± 9.3 years and for the diet group was 44.7 ± 9.4 years. Participants were screened to ensure that they fulfilled the inclusion criteria (described below).

Inclusion criteria

All participants in the Da Qing IGT and Diabetes Study (Pan 1997) lived in Da Qing, were over the age of 25 and received health care at one of the designated health clinics which were chosen

to participate in the study (roughly half of all Da Qing's clinics). Detailed inclusion criteria were not described.

The Oslo Diet and Exercise Study (Torjesen 1997) selected participants from the on-going screening programme who were relatively inactive (exercised once a week at the most and characterised by a maximal oxygen uptake [VO₂max] of 35.4 ± 5.9 ml/kg/min) as measured by a questionnaire, with a BMI greater than 24 kg/m², diastolic blood pressure of 86 to 99 mmHg, total cholesterol of 5.20 to 7.74 mmol/L, HDL-cholesterol of less than 1.20 mmol/L, and fasting triglycerides greater than 1.4 mmol/L at the initial screening. All criteria had to be fulfilled, as well as there being no obvious signs of cardiovascular disease or diabetes present, and that no drugs were being used that might have interfered with the accuracy of the test results.

Exclusion criteria

Participants were excluded from the Da Qing IGT and Diabetes Study if they were less than 25 kg/m² (Pan 1997). Further exclusion criteria were not described.

Participants were excluded if they had any symptoms of cardiovascular disease, or diabetes at the initial screening, or if unsuitable drugs were being used which could impede the test results (Torjesen 1997).

Diagnostic criteria

Pan (Pan 1997) used the 1985 WHO criteria (WHO 1985) to diagnose participants with diabetes by 2-hour glucose greater than 11.1 mmol/l or with fasting hyperglycaemia by fasting plasma glucose greater than 7.8 mmol/L.

Torjesen (Torjesen 1997) diagnosed participants with insulin resistance by using the following formulas:

- resistance = (insulin/7.2) / (22.5/glucose);
- relative β -cell function (%) = (20 x insulin/7.2) / (glucose-3.5);
- insulin was divided by 7.2 to obtain milli-units per litre from picomoles per litre.

Co-morbidities

During the 6-year follow-up of the Pan (Pan 1997) study, there were some fatalities reported, however, none were diabetes related. No co-morbidities were recorded for the Torjesen paper (Torjesen 1997).

Co-medications

No co-medications were recorded for the Torjesen (Torjesen 1997) or Pan (Pan 1997) studies.

Excluded studies

127 publications had to be excluded after careful evaluation of the full publication. Main reasons for exclusion were that the study was not a randomised control trial (n=88) or that the intervention was lifestyle and not diet alone (n=10) (for details see [Characteristics of excluded studies](#)).

Risk of bias in included studies

For details on methodological quality of included studies see [Appendix 2](#). Generally, the quality of the studies was quite poor. Risk of bias was assessed by the lead author only.

Allocation

Methods of randomisation were not described for the Da Qing IGT and Diabetes Study (Pan 1997). The methods of randomisation for the Oslo Diet and Exercise Study were not described, however, randomisation was stratified by gender (Holme 1993). Allocation concealment was not described in either paper.

Blinding

Method of blinding was not described in the Da Qing IGT and Diabetes Study (Pan 1997) paper. The Oslo Diet and Exercise Study was unmasked, but blinded for the objective blood analyses (Torjesen 1997).

Incomplete outcome data

All patients were screened initially and selected on the basis that they fulfilled all inclusion criteria. They were then randomised either by clinic (Pan 1997) or on an individual basis (Torjesen 1997). However, Pan (Pan 1997) reports that 47 out of the 577 original participants (8%) did not complete the study. Seven people refused follow-up, 29 left Da Qing in 1988 and 11 died from non-diabetic related illnesses. In the control group, there was one death from pneumonia and two from cirrhosis, and three deaths in the diet group (two from cancer, and one from septicaemia). There were no drop-out rates reported for the Torjesen paper (Torjesen 1997).

Definition of primary endpoint and secondary endpoints

Initially, the reviewers were studying the following primary outcomes: incidence of type 2 diabetes; glycaemic control measures (oral glucose tolerance test, both fasting and 2-hour and glycosylated haemoglobin; for the detection of impaired glucose tolerance); time to development or diagnosis of type 2 diabetes mellitus. The secondary outcomes that we wished to study were: quality of life (ideally, measured using a validated instrument); mortality; morbidity; weight; BMI; cost of intervention(s); serum cholesterol (LDL and/or HDL) and serum triglycerides; blood pressure; maximal exercise capacity and adverse effects. However, due to the

limitations of the data available in the papers which were available for inclusion, many of these endpoints could not be studied.

Power calculation

Power calculations were not performed for these studies.

Intention-to-treat and per-protocol analyses, missing data

Intention-to-treat, per-protocol analyses and procedures for missing data were not reported in these studies.

Compliance measures

Pan (Pan 1997) did not report any compliance measures, but it appeared that compliance was measured by attendance to clinics and by discussion with nurses and clinical staff.

Compliance data for the ODES were reported. At three and nine months, a blood sample was taken and analysed for HDL-cholesterol, triglycerides and total cholesterol. Each participant was also asked to respond to a 180 item food frequency questionnaire. The results of both were discussed with the participants to encourage compliance.

Funding

Sources of funding were not discussed in either paper.

Publication status

Both papers were published in *Diabetes Care*, a peer-reviewed journal.

Effects of interventions

From the included studies, no meta-analytic comparisons could be made due to insufficient information. However, data were inputted into the table to allow future comparisons to be made of one type of dietary advice versus another different type of dietary advice for a small number of outcomes including body mass index (BMI), weight and fasting glucose at a limited number of time points.

Baseline characteristics

For details of baseline characteristics see [Appendix 3](#).

For details of adverse events, primary and secondary outcomes see [Appendix 4](#), [Appendix 5](#) and [Appendix 6](#)

Dietary advice versus control

The two studies reported one type of dietary advice versus a control group which were provided with basic information. The Da Qing IGT and Diabetes Study (Pan 1997) randomised 163 participants to the two groups, and presented data at baseline and at the 6-year endpoint. The incidence of type 2 diabetes in the control group was 67.7% (95% confidence interval (CI) 59.8 to 75.2%) which was reduced to 43.8% (95% CI 35.5 to 54.7%) in the diet group. Overall, the dietary intervention group had a 33% reduction in the incidence of diabetes after 6 years ($P < 0.03$). Defined by WHO criteria (WHO 1985), the incidence of diabetes was 15.7/100 person-years (95% CI 12.7% to 18.7%) for the control group, and 10.0/100 (95% CI 7.5% to 12.5%) person-years in the diet group which was significantly different ($P < 0.05$). Fasting plasma glucose increased by 2.07 ± 0.82 mmol/L in the control group and 1.38 ± 0.81 mmol/L in the diet group. 2-hour plasma glucose increased by 3.96 ± 0.89 mmol/L in the control group and 1.48 ± 0.94 mmol/L in the diet arm. As dietary treatment and advice differed for participants of BMI less than 25kg/m^2 (lean) and participants with a BMI greater than 25kg/m^2 (overweight), some analyses were done to compare the two sub-groups. Incidence rates of diabetes in the control group of overweight participants were higher than incidence in the lean participants control group (17.2 versus 13.3/100 person years [$P < 0.05$]). In the lean participants, the incidence of developing diabetes was not significantly changed by the dietary intervention.

The Oslo Diet and Exercise Study (Torjesen 1997) presented data from 95 participants in two groups at baseline and at the 1-year endpoint. Many variables were measured at 0 and at 12 months with significant differences ($P < 0.05$) being found in change data measurements for the following (reported as diet group and control group respectively): insulin resistance -0.4 ± 0.2 versus 0.2 ± 0.2 ; fasting insulin (pmol/L) -7 ± 4 versus 4 ± 6 ; fasting C-peptide (pmol/L) -163 ± 49 versus -92 ± 53 ; fasting proinsulin (pmol/L) -2.2 ± 1.1 versus 1.6 ± 1.2 ; fasting blood glucose (mmol/L) -0.2 ± 0.1 versus 0.0 ± 0.1 ; BMI (kg/m^2) -1.3 ± 0.2 versus 0.4 ± 0.1 ; mBP (mm Hg) -5.2 ± 1.1 versus -1.5 ± 1.5 ; fasting triglycerides (mmol/L) -0.3 ± 0.2 versus -0.1 ± 0.3 ; fasting HDL-cholesterol (mmol/L) 0.06 ± 0.04 versus -0.05 ± 0.03 and PAI-1 (U/ml) 0.9 ± 3.8 versus 2.4 ± 3.3 . Weight was also measured (but not published), and noticeable differences were found between the diet group and the control group respectively at 12 months; weight (kg) -6.8 ± 19.5 versus 1.1 ± 13.8 although no significance level was published by the authors.

Incidence of type 2 diabetes

Pan (Pan 1997) reported incidence of diabetes for all groups of participants. As defined by WHO criteria (WHO 1985) the percentage of participants with 2-hour plasma glucose greater than 11.1mmol/L was 67.7% (95% CI 59.8% to 75.2%) for the control group and 43.8% (95% CI 35.5% to 54.7%) for the diet

group. Incidence of diabetes per 100 person-years was 15.7 (95% CI 12.7 to 18.7) for the control group and 10.0 (95% CI 7.5 to 12.5) for the diet group which was a significant reduction for the intervention ($P < 0.05$).

Time to development or diagnosis of type 2 diabetes mellitus was not measured in either study.

Glycaemic control measures

Pan (Pan 1997) also reported the incidence of fasting plasma glucose greater than 7.8mmol/L which was 41.4% for the control group and 16.2% for the dietary intervention group. Incidence of fasting hyperglycemia per 100 person-years was 9.6 (95% CI 7.2 to 12.0) for the control group, and 3.7 (95% CI 2.1 to 5.3) for the diet group. Fasting plasma glucose increased by 2.07 ± 0.82 mmol/L in the control group and 1.38 ± 0.81 mmol/L in the diet group. 2-hour plasma glucose increased by 3.96 ± 0.89 mmol/L in the control group and 1.48 ± 0.94 mmol/L in the diet arm.

Torjesen (Torjesen 1997) describes only change data for fasting glucose (mmol/L), which was -0.2 ± 0.1 and 0.0 ± 0.1 and for post-oral glucose glucose (mmol/L) which was -0.3 ± 0.2 and -0.2 ± 0.3 for the diet group and control groups, respectively.

Other outcomes

Pan (Pan 1997) reported no additional or secondary outcomes. In the Oslo Diet and Exercise Study (Torjesen 1997) weight was reduced noticeably by -6.8 ± 19.5 kg in the diet group compared to an increase of 1.1 ± 13.8 in the control group. Significant differences ($P < 0.05$) were also reported for BMI (kg/m^2) -1.3 ± 0.2 for diet group versus 0.4 ± 0.1 for control group; mean blood pressure (mBP [mmHg]) -5.2 ± 1.1 for diet versus -1.5 ± 1.5 for control; fasting triglycerides (mmol/L) -0.3 ± 0.2 for diet versus -0.1 ± 0.3 for control; fasting HDL-cholesterol (mmol/L) 0.06 ± 0.04 for diet versus -0.05 ± 0.03 for control and PAI-1 (U/ml) 0.9 ± 3.8 for diet versus 2.4 ± 3.3 for control group. The aim of the Oslo Diet and Exercise Study (Torjesen 1997) was to assess the effect of diet and exercise, either as single interventions, or in combination, on the development of insulin resistance. The calculated relative insulin resistance varied widely in participants. However, despite the large variation, the diet intervention lowered the mean insulin resistance after 12 months from 4.6 to 4.2. Other significant insulin-related changes ($P < 0.05$) were fasting insulin (pmol/L) -7 ± 4 for diet versus 4 ± 6 for control; fasting C-peptide (pmol/L) -163 ± 49 for diet versus -92 ± 53 for control; fasting proinsulin (pmol/L) -2.2 ± 1.1 for diet versus 1.6 ± 1.2 for control and overall insulin resistance being -0.4 ± 0.2 for dietary intervention and 0.2 ± 0.2 for control.

Quality of life, mortality, morbidity, cost of intervention, maximal exercise capacity and adverse effects were not reported by this study.

Heterogeneity

Due to the lack of data, tests for heterogeneity could not be performed.

Subgroup analyses

Subgroup analyses were planned, however, due to the lack of data they could not be carried out. However, Pan (Pan 1997) did report data for lean participants (BMI less than $25\text{kg}/\text{m}^2$) and overweight participants (BMI greater than $25\text{kg}/\text{m}^2$) which would be interesting analyses to follow up.

Sensitivity analyses

No sensitivity analyses were performed.

Publication and small study bias

Not performed due to small number of studies.

DISCUSSION

Summary

This Cochrane review examined five papers, reporting data from two large diabetes prevention trials. These were the Da Qing IGT and Diabetes Study, and the Oslo Diet and Exercise Study, where dietary advice versus control group data were reviewed for 358 participants. Due to the small amount of data available, no meta-analyses could be carried out. However, it appears that diet can have a significant impact on risk factors for the prevention of diabetes, compared to a control group over a 12 month period. The Oslo Diet and Exercise Study (Torjesen 1997) found that many variables changed significantly ($P < 0.05$) and beneficially including insulin resistance, fasting insulin (pmol/L), blood glucose and plasma lipoprotein profiles and anthropometric measurements such as weight (kg) and body mass index (kg/m^2).

Over the six year study, Da Qing IGT and Diabetes Study (Pan 1997) found a 33% reduction in the incidence of diabetes ($P < 0.03$) for participants in the diet group, compared to the control group. The only similar data that were available for the two studies related to fasting plasma glucose measurements. Fasting plasma glucose changed by 2.07 ± 0.82 mmol/L in the control group compared to 1.38 ± 0.81 mmol/L in the diet group after six years in the Da Qing IGT and Diabetes study (Pan 1997). Fasting plasma glucose changed by -0.2 ± 0.1 mmol/L in the diet group versus 0.0 ± 0.1 mmol/L in the control group after one year in the Oslo Diet and Exercise Study (Torjesen 1997). However, due to the varying time-points at which measurements were taken, more information

would be required before a comparison of the two different types of dietary advice can be carried out.

Limitations of the review

The main limitation of this review is the lack of studies and available related data. Despite the current situation we are facing with the diabetes epidemic, there are not enough long-term data available to come to any confident conclusions.

AUTHORS' CONCLUSIONS

Implications for practice

Although only two types of dietary advice were provided in the trials, common factors were reduction of energy intake and simple sugars, an increase in fresh fruit and vegetables. Taking this into account, diets which are similar to those prescribed in the studies, alongside the frequent contact with dietary advisors, appear to have a positive effect on minimising the risk factors commonly associated with the development of diabetes.

Implications for research

Additional research needs to be carried out into the best type of diet, and the optimal frequency and type of contact with dietary

advisors, to maximise participant compliance to any prescribed dietary treatments.

Conclusions

Although more evidence is required, the data which are available do suggest that there are benefits in following an energy-controlled diet with an increase in consumption of fresh fruit and vegetables, and a decrease in simple sugars intake. However, another overriding factor is the frequency of support and guidance provided by the dietary advisors, which occurred at least every 3 to 6 months in both studies, encouraging compliance to the prescribed diets. More well-designed, long-term studies, providing well-reported, high-quality data are required before conclusions can be made with confidence into the best dietary advice to give to high-risk patients for the prevention of diabetes mellitus in adults.

ACKNOWLEDGEMENTS

Barbara V. Howard, USA for providing correspondence regarding the Pan study.

Boyd Swinburn, Australia for providing correspondence regarding his study.

Peter A. Torjesen, Norway for providing unpublished data for his study.

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* Indicates the major publication for the study

CHARACTERISTICS OF STUDIES

Characteristics of included studies [ordered by study ID]

Pan 1997

Methods	110,660 participants from 33 clinics in Da Qing, China were screened for IGT and Type 2 diabetes. Using the WHO criteria, 577 were classified as having IGT and were randomized by clinic to the trial, as a control group or to one of three intervention groups- diet only, exercise only or diet and exercise. Follow-up evaluations were conducted at 2-year intervals over a 6-year period to identify individuals who developed NIDDM. In this review, interest is in the diet only vs control arms of the study Assessment category: C (two points awarded) - high risk of bias.
Participants	577 participants were randomised by clinic to the groups. Diet only intervention (45% male, 55% female) mean age 44.7 years, and control group (55% male, 45% female), mean age 46.5 years.
Interventions	The diet only intervention consisted of dietary advice and prescription of diets. Advice included increasing vegetable consumption and reducing the intake of alcohol and simple sugars. The diet was to consist of 55% to 65% carbohydrates, 10% to 15% protein and 25% 30% fat. This was provided during group sessions and through individual counselling.
Outcomes	At 2-year intervals, a systematic evaluation of each participant was carried out recording diet and exercise changes and individual advice on intervention adherence. Height, weight and blood pressure was measured alongside 2-hour fasting blood glucose. Compared to the control, there was a 33% overall reduction of the incidence of diabetes in the diet only group ($P < 0.03$)
Notes	

Torjesen 1997

Methods	An unmasked, randomized, 2x2 factorial intervention was carried out with a duration of one year. Patients were allocated to diet, diet plus exercise, exercise and control groups. For the purpose of this study, focus was on diet only vs control Assessment category: C (three points awarded) - high risk of bias.
Participants	There were 219 subjects with diastolic blood pressure 86 to 99 mmHg, HDL-cholesterol < 1.2 mmol/L, total cholesterol of 5.20 to 7.74 mmol/L, and BMI $>$ greater than 24 kg/m ² . there were 55 participants in the diet group, and 43 in the control group
Interventions	The diet included increased intake of fish and reduced total fat intake.
Outcomes	After 1 year, the diet intervention significantly reduced BMI by 1.3 kg ($P < 0.0001$). Mean BMI increased by 0.4 kg/m ² during the intervention period in the control group.
Notes	

HbA1c = Glycosylated haemoglobin A1c

BMI = Body mass index

CG = Control group
IG = Intervention group
NDDG = National Diabetes Data Group
WHO = World Health Organisation
Unless specified, ## ± ## is mean ± SD

Characteristics of excluded studies *[ordered by study ID]*

Adam-Perrot 2006	i) not an RCT
Aguillar 1999	i) not an RCT
Angelo 2005	i) not an RCT
Arky 1983	i) not an RCT
Assal 1997	i) not an RCT
Astrup 2001	i) not an RCT
Avenell 2004	i) not an RCT
Barnard 1994	i) not an RCT
Bazzano 2005	i) not an RCT
Bell 2000	i) not an RCT
Bennett 1997	i) not an RCT
Bhoraskar 2005	i) not an RCT
Bloomgarden 1998	i) not an RCT
Bloomgarden 2004	i) not an RCT
Ciliska 1995	i) not an RCT
Clark 1991	i) not an RCT
Connor 2003	i) not an RCT
Costacou 2003	i) not an RCT
Darnton-Hill 2004	i) not an RCT
Davey-Smith 2005	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
de Leiva 1998	i) not an RCT

(Continued)

Doherty 2006	i) not an RCT
Dyson 1997	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Dyson 2004	i) not an RCT
Enas 2003	i) not an RCT
Entrala-Bueno 2003	i) not an RCT
Eriksson 1991	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Eriksson 1999	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Eschwege 2000	i) not an RCT
Eyre 2004	i) not an RCT
Fernandez-Soto 1990	i) not an RCT
Feskens 1999	i) not an RCT
Florez 1997	i) not an RCT
Franz 2003	i) not an RCT
Fujimoto 2000	i) not an RCT
Gin 2003	i) not an RCT
Goldberg 1998	i) not an RCT
Gruber 2006	i) not an RCT
Haffner 2002	i) not an RCT
Hamdy 2001	i) not an RCT

(Continued)

Hamman 2006	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Inzucchi 2005	i) not an RCT
Jain 2001	i) not an RCT
Kanaya 2003	i) not an RCT
Klein 2004a	i) not an RCT
Klein 2004b	i) not an RCT
Knowler 1995	i) not an RCT
Knowler 2002	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Kriska 1994	i) not an RCT
Kriska 2004	i) not an RCT
Laakso 2005	i) not an RCT
Laaksonen 2005	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Lamonte 2005	i) not an RCT
Lean 2003	i) not an RCT
Liao 2002	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Liberopoulos 2006	i) not an RCT
Lindahl 1999	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was intervention

(Continued)

	v) weight loss measured vi) no measure of diabetic control
Lindstrom 2003	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Lindstrom 2005	i) not an RCT
Maggio 1997	i) not an RCT
Mann 2006a	i) not an RCT
Mann 2006b	i) not an RCT
Manson 1994	i) not an RCT
McCarty 2000	i) not an RCT
Muller 2004	i) not an RCT
Norris 2005a	i) not an RCT
Norris 2005b	i) not an RCT
Panzer 2003	i) not an RCT
Pfohl 2001	i) not an RCT
Pittas 2003	i) not an RCT
Pospisilova 2006	i) not an RCT
Quinn 2003	i) not an RCT
Riddle 1997	i) not an RCT
Ruderman 1990	i) not an RCT
Ruhe 2003	i) not an RCT
Ryan 2003	i) not an RCT
Sartorelli 2006	i) not an RCT
Satterfield 2003	i) not an RCT

(Continued)

Scherthaner 2003	i) not an RCT
Schonthal 1999	i) not an RCT
Schulze 2005	i) not an RCT
Schwarz 2006	i) not an RCT
Simpson 2003	i) not an RCT
Steyn 2004	i) not an RCT
Thompson 2001	i) not an RCT
Toeller 2005	i) not an RCT
Tuomilehto 1992	i) not an RCT
Tuomilehto 2001	i) is an RCT ii) follow up >12 months iii) participants >18 years old iv) dietary advice was not the intervention
Uusitupa 2005	i) not an RCT
Uutela 2004	i) not an RCT
Venkat Narayan 2003	i) not an RCT
Vidal 2005	i) not an RCT
Vijgen 2006	i) not an RCT
Warnken 2005	i) not an RCT
Weickert 2005	i) not an RCT
Williamson 2004	i) not an RCT
Wylie-Rosett 2006	i) not an RCT
Yamaoka 2005	i) not an RCT
Zeman 2005	i) not an RCT

NB1. Any “no” disqualifies study from inclusion.

NB2. If excluded because they are “treatment studies”, these have been included in a previously published treatment review.

DATA AND ANALYSES

Comparison 1. Dietary Advice vs Control

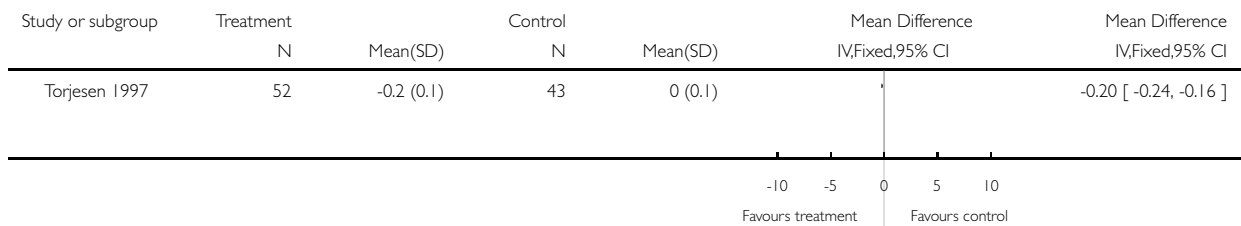
Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Fasting blood glucose (mmol/l) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
2 Insulin resistance at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
3 % B-cell function at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
4 Development of diabetes	0		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
4.1 % developing diabetes (defined by WHO criteria [2-hour plasma glucose >11.1mmol/l]) at 6 years	0		Mean Difference (IV, Fixed, 95% CI)	Not estimable
4.2 Incidence of diabetes (defined using WHO criteria) per 100 person years at 6 years	0		Mean Difference (IV, Fixed, 95% CI)	Not estimable
5 Fasting C-peptide (pmol/l) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
6 Fasting blood glucose (mmol/l) at 6 years	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
7 Fasting hyperglycemia	0		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
7.1 defined using fasting glucose >140mg/dl [7.8mmol] per 100 person years at 6 years	0		Mean Difference (IV, Fixed, 95% CI)	Not estimable
7.2 % incidence of fasting glucose >140mg/dl [7.8mmol/l]	0		Mean Difference (IV, Fixed, 95% CI)	Not estimable
8 Fasting proinsulin (pmol/l) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
9 Post-oral glucose glucose (mmol/l) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
10 Fasting triglycerides (mmol/l) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
11 Fasting HDL-cholesterol (mmol/l)	1	95	Mean Difference (IV, Fixed, 95% CI)	0.11 [0.10, 0.12]
12 PAI-1 (U/ml) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
13 2-hour blood glucose (mmol/l) at 6 years	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
14 mBP (mmHg) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
15 BMI (kg/m2) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
16 Weight (kg) at 1 year	0		Mean Difference (IV, Fixed, 95% CI)	Totals not selected
17 Fasting insulin (pmol/l) at 1 year	1		Mean Difference (IV, Fixed, 95% CI)	Totals not selected

Analysis 1.1. Comparison 1 Dietary Advice vs Control, Outcome 1 Fasting blood glucose (mmol/l) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 1 Fasting blood glucose (mmol/l) at 1 year

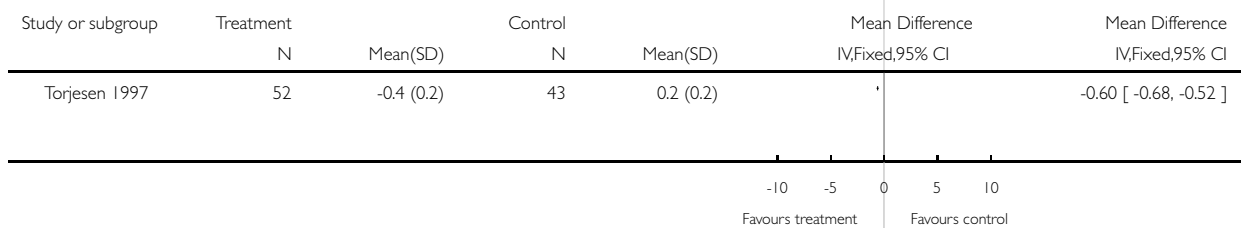


Analysis 1.2. Comparison 1 Dietary Advice vs Control, Outcome 2 Insulin resistance at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 2 Insulin resistance at 1 year

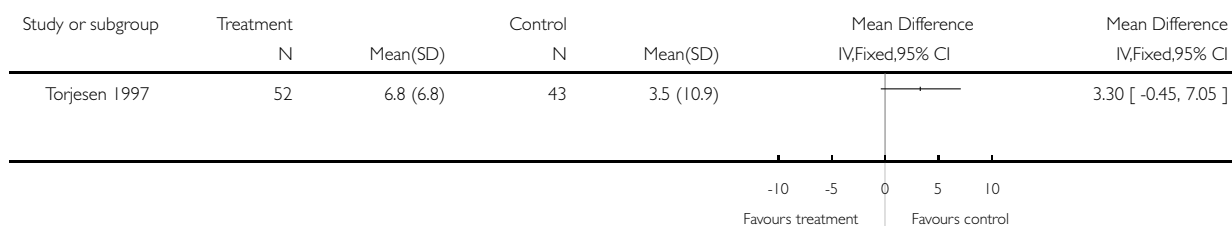


Analysis 1.3. Comparison 1 Dietary Advice vs Control, Outcome 3 % B-cell function at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 3 % B-cell function at 1 year

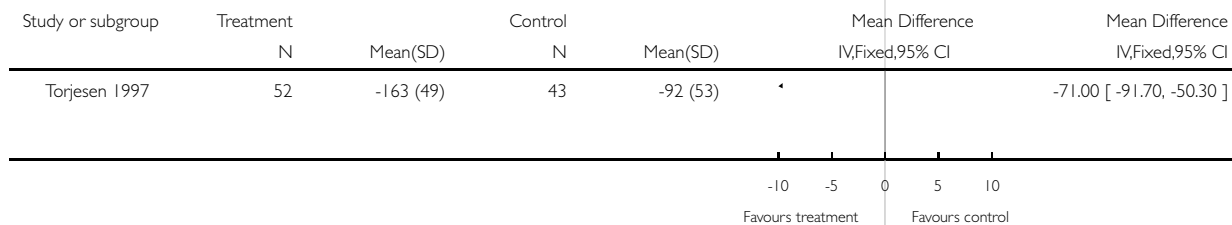


Analysis 1.5. Comparison 1 Dietary Advice vs Control, Outcome 5 Fasting C-peptide (pmol/l) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 5 Fasting C-peptide (pmol/l) at 1 year

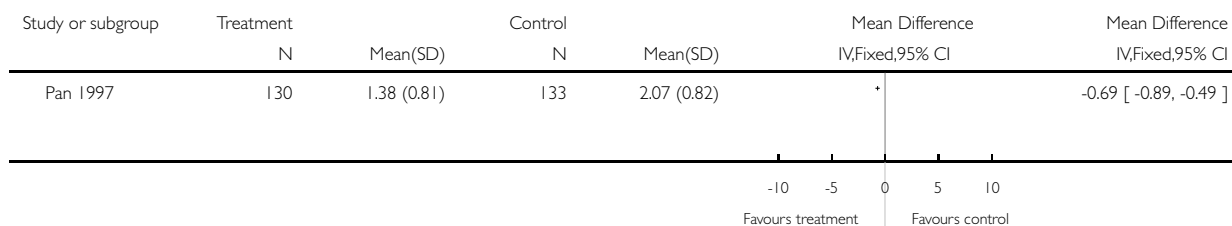


Analysis 1.6. Comparison 1 Dietary Advice vs Control, Outcome 6 Fasting blood glucose (mmol/l) at 6 years.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 6 Fasting blood glucose (mmol/l) at 6 years

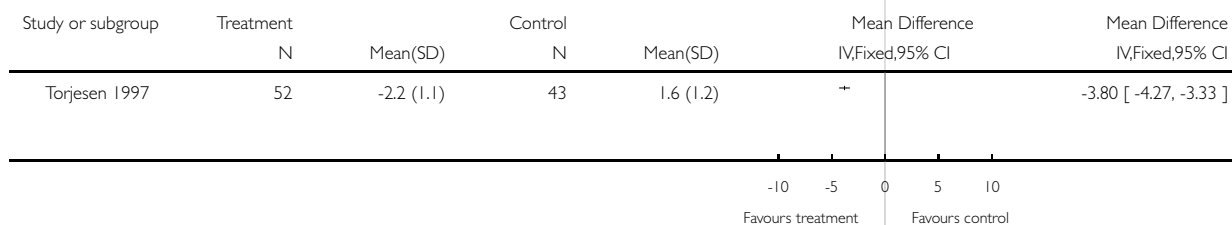


Analysis 1.8. Comparison 1 Dietary Advice vs Control, Outcome 8 Fasting proinsulin (pmol/l) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 8 Fasting proinsulin (pmol/l) at 1 year

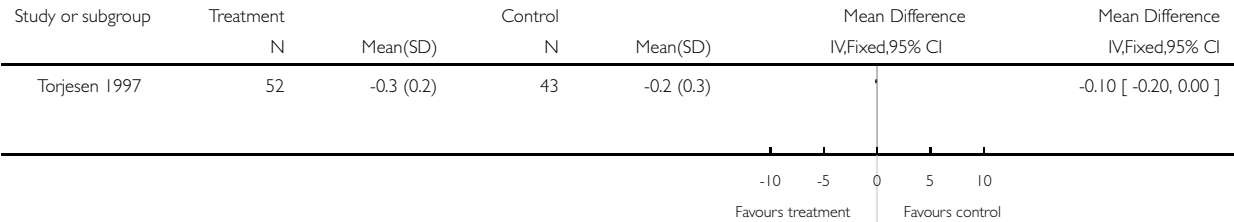


Analysis 1.9. Comparison 1 Dietary Advice vs Control, Outcome 9 Post-oral glucose glucose (mmol/l) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 9 Post-oral glucose glucose (mmol/l) at 1 year

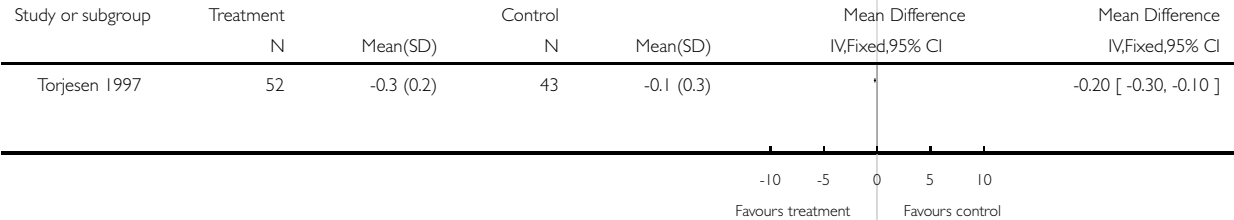


Analysis 1.10. Comparison 1 Dietary Advice vs Control, Outcome 10 Fasting triglycerides (mmol/l) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 10 Fasting triglycerides (mmol/l) at 1 year

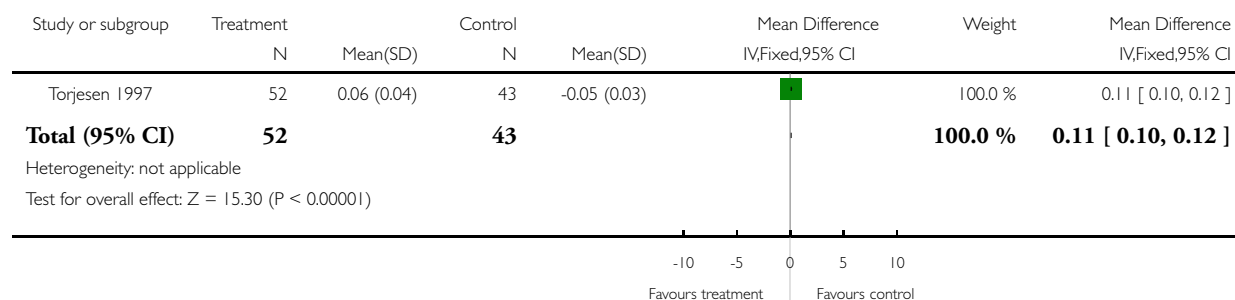


Analysis 1.11. Comparison 1 Dietary Advice vs Control, Outcome 11 Fasting HDL-cholesterol (mmol/l).

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 11 Fasting HDL-cholesterol (mmol/l)

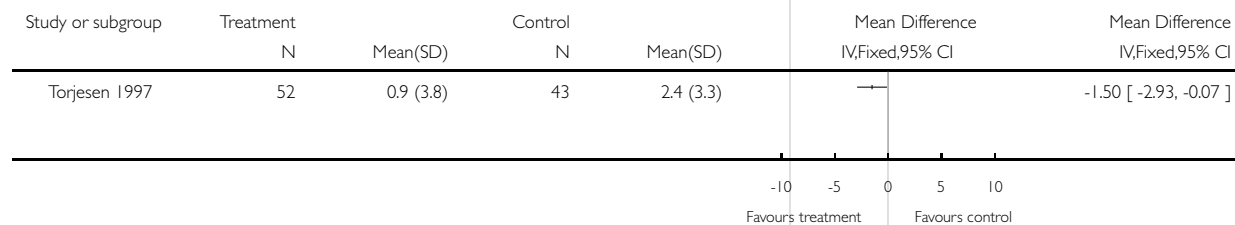


Analysis 1.12. Comparison 1 Dietary Advice vs Control, Outcome 12 PAI-I (U/ml) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 12 PAI-I (U/ml) at 1 year

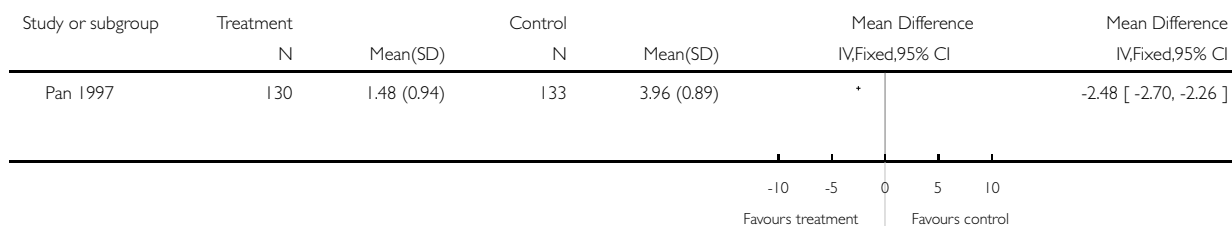


Analysis 1.13. Comparison 1 Dietary Advice vs Control, Outcome 13 2-hour blood glucose (mmol/l) at 6 years.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 13 2-hour blood glucose (mmol/l) at 6 years

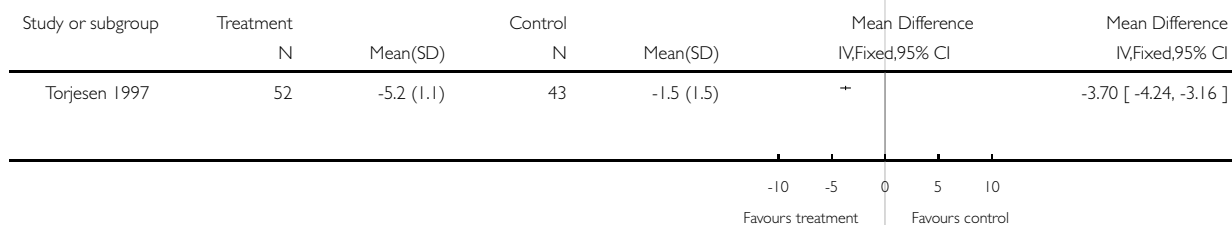


Analysis 1.14. Comparison 1 Dietary Advice vs Control, Outcome 14 mBP (mmHg) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 14 mBP (mmHg) at 1 year

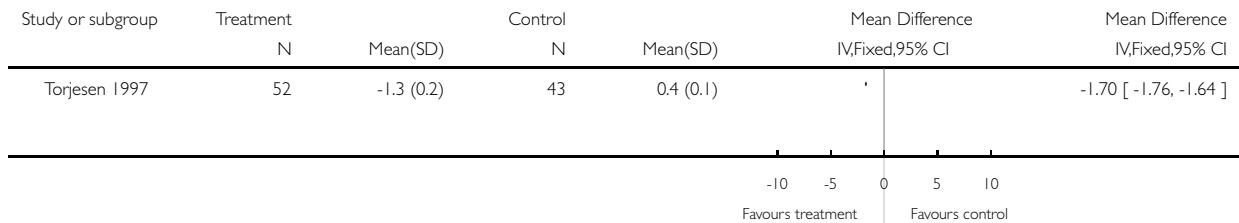


Analysis 1.15. Comparison 1 Dietary Advice vs Control, Outcome 15 BMI (kg/m2) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 15 BMI (kg/m2) at 1 year

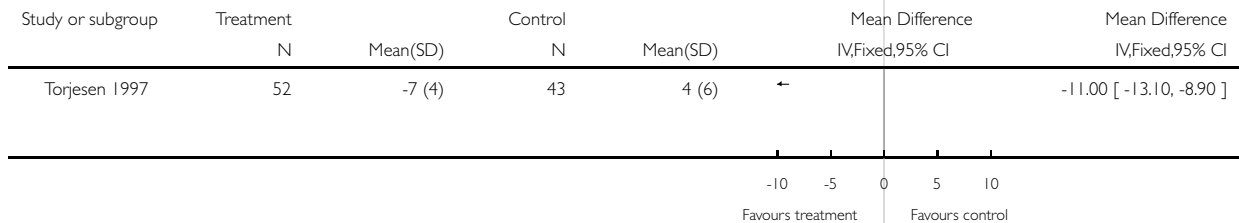


Analysis 1.17. Comparison 1 Dietary Advice vs Control, Outcome 17 Fasting insulin (pmol/l) at 1 year.

Review: Dietary advice for the prevention of type 2 diabetes mellitus in adults

Comparison: 1 Dietary Advice vs Control

Outcome: 17 Fasting insulin (pmol/l) at 1 year



APPENDICES

Appendix 1. Search strategy

Search terms

NOTE: Unless otherwise stated, search terms are free text terms; MeSH: Medical subject heading (MEDLINE medical index term); a dollar sign stands for 'any character(s)' and a question mark stands for an 'optional character'. This search was created for use with the OVID gateway.

(Continued)

Adj: The adjacency operator (adjn) retrieves two or more query terms within n words of each other, (any order). The number n may be any number from 1 through 99, and immediately follows adj without a space, as in adj7, adj3 etc.

Explode: In order to search for all the terms that are more specific than the original term, as well as the original term, you 'explode' that term. For example, if you explode the term 'Wounds and Injuries', your search will include all the specific types of wounds such as burns, contusions, and fractures.

TYPE 2 DIABETES MELLITUS

1. exp diabetes mellitus, Type II/
2. exp insulin resistance/
3. impaired glucose toleranc\$.tw.
4. glucose intoleranc\$.tw.
5. insulin\$ resistanc\$.tw.
6. exp obesity in diabetes/
7. (obes\$ adj diabet\$).tw.
8. (MODY or NIDDM).tw.
9. (non insulin\$ depend\$ or noninsulin\$ depend\$ or noninsulin?depend\$ or non insulin?depend\$).tw.
10. ((typ\$ 2 or typ\$ II) adj diabet\$).tw.
11. ((keto?resist\$ or non?keto\$) adj diabet\$).tw.
12. ((adult\$ or matur\$ or late or slow or stabl\$) adj diabet\$).tw.
13. (insulin\$ defic\$ adj relativ\$).tw.
14. pluri?metabolic\$ syndrom\$.tw.
15. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14
16. exp diabetes insipidus/
17. diabet\$ insipidus.tw.
18. 16 or 17
19. 15 not 18

DIET INTERVENTIONS

20. explode Diet Therapy/ [MeSH, all subheadings]
21. Explode Diet/ [MeSH, all subheadings]
22. Nutrition/[MeSH, all subheadings]
23. (diet\$ adj5 diabet\$).ab,ti.
24. (diet\$ adj5 carbohydrat\$).ab,ti.
25. (diet\$ adj5 fat\$).ab,ti.
26. (diet\$ adj5 weigh\$).ab,ti.
27. (diet\$ adj5 sugar\$).ab,ti.
28. (diet\$ adj5 glyce?em\$).ab,ti.
29. 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28

SYSTEMATIC REVIEWS/META-ANALYSIS

30. exp meta-analysis/
31. exp Review Literature/
32. meta-analysis.pt.
33. review.pt.
34. 30 or 31 or 32 or 33
35. letter.pt.
36. comment.pt.

(Continued)

- 37. editorial.pt.
- 38. historical-article.pt.
- 39. 35 or 36 or 37 or 38
- 40. 34 not 39
- 41. ((systematic\$ or quantitativ\$ or methodologic\$)adj (review\$ or overview\$)).tw.
- 42. meta?anal\$.tw.
- 43. (integrativ\$ research review\$ or research integration\$).tw.
- 44. quantitativ\$ synthes\$.tw.
- 45. (pooling\$ or pooled analys\$ or mantel\$ haenszel\$).tw.
- 46. (peto\$ or der?simonian\$ or fixed effect\$ or random effect\$).tw.
- 47. 41 or 42 or 43 or 44 or 45 or 46
- 48. 40 or 47
- 49. limit 48 to human

RANDOMISED CONTROLLED TRIALS

- 50. randomized controlled trial.pt.
- 51. randomized controlled trials/[MeSH, all subheadings]
- 52. controlled clinical trial.pt.
- 53. random allocation/[MeSH, all subheadings]
- 54. double blind method/[MeSH, all subheadings]
- 55. single-blind method/[MeSH, all subheadings]
- 56. 50 or 51 or 52 or 53 or 54 or 55
- 57. clinical trial.pt.
- 58. explode clinical trials/[MeSH, all subheadings]
- 59. (clin\$ adj5 trial\$).tw.
- 60. ((singl\$ or doubl\$ or trebl\$ or tripl\$) adj5 (blind\$ or mask\$)).tw.
- 61. placebos/[MeSH, all subheadings]
- 62. placebo\$.tw.
- 63. random\$.tw.
- 64. research design/[MeSH, all subheadings]
- 65. 57 or 58 or 59 or 60 or 61 or 62 or 63 or 64
- 66. comparative study/[MeSH, all subheadings]
- 67. explode evaluation studies/[MeSH, all subheadings]
- 68. follow up studies/[MeSH, all subheadings]
- 69. prospective studies/[MeSH, all subheadings]
- 70. (control\$ or prospectiv\$ or volunteer\$).tw.
- 71. 66 or 67 or 68 or 69 or 70
- 72. animal/ not (human/ and animal/)
- 73. 56 or 65 or 71
- 74. 73 not 72

TYPE 2 DIABETES AND DIETARY INTERVENTIONS

- 75. 19 and 29

TYPE 2 DIABETES AND DIETARY INTERVENTIONS AND RANDOMISED CONTROLLED TRIALS

- 76. 75 and 74

TYPE 2 DIABETES AND DIETARY INTERVENTIONS AND SYSTEMATIC REVIEW/META-ANALYSES

- 53. 75 and 49

(Continued)

The MEDLINE search will be run as above, with the randomised controlled trials and systematic reviews searches added on separately.

The CINAHL search will be run as above, with randomised controlled trials search added on separately.

The AMED search will be run as above, with randomised controlled trials search added on separately.

The EMBASE search will be adapted from the original search above.

Appendix 2. Study quality

Characteristic	Pan 1997	Torjesen 1997
Intervention 1 (I1) / intervention 2 (I2) / control 1 (C1)	I1: Dietary Advice C1: Control	I1: Dietary Advice C1: Control
Randomised controlled clinical trial (RCT)	Y	Y
Non-inferiority / equivalence trial	?	?
Controlled clinical trial	Y	Y
Design: parallel, crossover, factorial RCT	?	Y
Design: crossover study	?	Y
Design: factorial study	?	Y
Crossover study: wash-out phase	?	?
Crossover study: carryover effect tested	?	?
Crossover study: period effect tested	?	?
Method of randomisation (specify)	By Clinic	Stratified by gender
Unit of randomisation (individuals, cluster - specify)	Cluster	Individuals
Randomisation stratified for centres	Y	N
Randomisation ratio	?	?
Concealment of allocation (specify)	N	N

(Continued)

Stated blinding (open; single, double, triple blind)	?	?
Actual blinding: participant	N	N
Actual blinding: caregiver / treatment administrator	?	?
Actual blinding: outcome assessor	?	Y
Actual blinding: others	?	?
Blinding checked: participant	?	?
Blinding checked: caregiver / treatment administrator	?	?
Primary endpoint defined	N	N
[n] of primary endpoint(s)	2	12
[n] of secondary endpoints	3	?
Total [n] of endpoints	5	12
Prior publication of study design	Y	Y
Outcomes of prior and current publication identical	Y	Y
Power calculation	?	?
[n] participants per group calculated	?	?
Non-inferiority trial: interval for equivalence specified	?	?
Intention-to-treat analysis (ITT)	?	?
Per-protocol-analysis	Y	Y
ITT defined	?	?
Missing data: last-observation-carried-forward (LOCF)	?	?
Missing data: other methods	?	?

(Continued)

LOCF defined	?	?
[n] of screened participants (I1/ I2 / C1 / total)	110,660	26,000
[n] of randomised participants (for primary endpoint)	Total: 1207 (in all 4 groups)	Total: 209 (in all 4 groups) 98 (in the 2 groups studied)
[n] of participants finishing the study	577 (total of 4 groups) 169 (in the 2 groups studied)	Total: 209 (in all 4 groups) 98 (in the 2 groups studied)
[n] of patients analysed (for primary end-point)	530 (total of 4 groups) 163 (in the 2 groups studied)	Total: 209 (in all 4 groups) 98 (in the 2 groups studied)
Description of discontinuing participants	Seven people refused follow-up, 29 left Da Qing in 1988 and eleven died from non-diabetic related illnesses. In the control group, there was one from pneumonia and two from cirrhosis, and three in the diet group (two from cancer, and one from septicemia).	
Drop-outs (reasons explained)	7 refused follow-up	
Withdrawals (reasons explained)	29 left Da Qing in 1988	
Losses-to-follow-up (reasons explained)	11 died (in total of 4 groups) 6 died (in 2 groups studied)	
[n] of participants who discontinued	47	
[%] discontinuation rate	8%	
Discontinuation rate similar between groups	Y	
[%] crossover between groups	?	
Differences [n] calculated to analysed patients	?	?
Adjustment for multiple outcomes / repeated measurements	?	?
Baseline characteristics: clinically relevant differences	?	?

(Continued)

Treatment identical (apart from intervention)	Y	?
Compliance measured	?	Y
Other important covariates measured (specify)	?	?
Co-morbidities measured	?	?
Co-medications measured	?	?
Specific doubts about study quality		
Funding: commercial	?	?
Funding: non-commercial	?	?
Publication status: peer review journal	Y	Y
Publication status: journal supplement	N	N
Publication status: abstract	N	N
Publication status: other	N	N
Notes		
Symbols & abbreviations: Y = yes; N = no; ? = unclear I = intervention; C = control		

Appendix 3. Baseline characteristics

Characteristic	Torjesen (1997)	Pan (1997)
Dietary Intervention 1 / control 1 (C1)	<p>I1: increase of fish and reduced total fat intake</p> <p>C1: no prescribed dietary advice</p>	<p>I1: A diet was prescribed containing 25-30 kcal/kg body weight, 55-65% carbohydrate, 10-15% protein, and 25-30% fat. Encouraged to eat more vegetables, control alcohol and reduce intake of simple sugars</p> <p>C1: General information and brochures regarding general instruction for diet and increasing physical activity.</p>

(Continued)

[n] (I1/ C1 / total)	I1: 52 C1: 43 Total: 95	I1: 130 C1: 133 Total: 263
Sex M/F [n,%]	I1: not stated C1: not stated	I1: 59/71 C1: 73/60
Age [years] mean (SD)	I1: not stated C1: not stated Total:	I1: 44.7 (9.4) C1: 46.5 (9.3)
Ethnic groups [%]	not stated	not stated
Insulin Resistance	I1: 4.60 (2.10) C1: 4.89 (4.64)	
Weight [kg] mean (SD)	I1: 92.73 (19.48) C1: 89.33 (13.77)	
Body mass index [kg/m2] mean (SD)	I1: 29.7 (4.2) C1: 28.3 (3.1)	I1: 25.3 (3.80) C1: 26.2 (3.9)
B-cell function [%] mean (SD)	I1: 179 (71) C1: 207 (107)	
Fasting Insulin [pmol/l] mean (SD)	I1: 130 (52) C1: 142 (108)	
Fasting C-Peptide [pmol/l] mean (SD)	I1: 1073 (473) C1: 1111 (686)	
Fasting proinsulin [pmol/l] mean (SD)	I1: 14 (11) C1: 15 (22)	
Fasting Glucose [mmol/l] mean (SD)	I1: 5.65 (0.81) C1: 5.41 (0.52)	I1: 5.56 (0.81) C1: 5.52 (0.82)

(Continued)

Post-oral glucose glucose [mmol/l] mean (SD)	I1: 8.45 (2.52) C1: 7.85 (2.20)	
2 hour glucose [mmol/l] mean (SD)		I1: 9.03 (0.94) C1: 9.03 (0.89)
mBP [mmHg] mean (SD)	I1: 100 (10) C1: 98 (8)	
Fasting Triglycerides [mmol/l] mean (SD)	I1: 2.39 (1.35) C1: 2.29 (0.84)	
Fasting HDL Cholesterol [mmol/l] mean (SD)	I1: 0.93 (0.22) C1: 1.05 (0.17)	
PAI-1 [U/ml] mean (SD)	I1: 22 (17) C1: 18 (14)	
Notes		
Symbols & abbreviations: Y = yes; N = no; ? = unclear I = intervention; C = control		

Appendix 4. Adverse events

Characteristic	Pan 1997	Torjesen 1997
Intervention 1 (I1) / intervention 2 (I2) / control 1 (C1)	I1: 130 C: 133	I1: 55 C: 43
[n] of participants who died	11	?
[n] adverse events (I1/ I2 / C1 / total)	I1: 3 C: 3	?
[%] adverse events	8%	?

(Continued)

[n] serious adverse events	?	?
[%] serious adverse events	?	?
[n] drop-outs due to adverse events	I1: I2: C1: Total:	?
[%] drop-outs due to adverse events	?	?
[n] hospitalisation	?	?
[%] hospitalisation	?	?
[n] out-patient treatment	?	?
[%] out-patient treatment	?	?
[n] hypoglycaemic episodes	?	?
[%] hypoglycaemic episodes	?	?
[n] severe hypoglycaemic episodes	?	?
[%] severe hypoglycaemic episodes	?	?
[n] nocturnal hypoglycaemic episodes	?	?
[%] nocturnal hypoglycaemic episodes	?	?
[n] with symptoms	?	?
[%] with symptoms	?	?
Symbols & abbreviations: Y = yes; N = no; ? = unclear I = intervention; C = control		

Appendix 5. Primary outcome data

Study	All-cause mortality	Morbidity	Quality of life
Pan 1997: I1: Dietary Advice C: Control	I1	?	?
Torjesen 1997: I1: Dietary Advice C: Control	?	?	?
Symbols & abbreviations: ? = unclear; I = intervention; C = control			

Appendix 6. Secondary outcome data

Characteristic	HbA1c (mean [SD])
Pan 1997: I1: C1:	I1: I2: C1:
Torjesen 1997: I1: C1:	I1: I2: C1: Total:
Symbols & abbreviations: ? = unclear; I = intervention; C = control	

WHAT'S NEW

Last assessed as up-to-date: 31 October 2007.

5 May 2008	Amended	Converted to new review format.
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HISTORY

Protocol first published: Issue 1, 2005

Review first published: Issue 3, 2008

CONTRIBUTIONS OF AUTHORS

LUCIE NIELD: co-ordinating the review, data collection for the review, undertaking searches, organising retrieval of papers, screening retrieved papers against inclusion criteria, writing to authors of papers for additional information, providing additional data about papers, obtaining and screening data on unpublished studies, abstracting data from papers, data management of the review, entering data into RevMan, analysis of data, writing the review.

HELEN MOORE: designing the review protocol, conceiving the review, developing search strategy, screening retrieved papers against inclusion criteria, appraising quality of papers, abstracting data from papers, data management for the review.

CAROLYN SUMMERBELL: conceiving the review, designing the review, screening search results, screening retrieved papers against inclusion criteria, appraising quality of papers, abstracting data from papers, obtaining and screening data on unpublished studies, interpretation of data, providing general advice on the review, securing funding for the review, performed previous work that was the foundation of current study.

LEE HOOPER: screening of retrieved papers against inclusion criteria, appraising quality of papers, abstracting data from papers, providing a clinical perspective, performed previous work that was the foundation of the current study.

VICKI WHITTAKER: data management for the review, analysis of data, interpretation of data.

DECLARATIONS OF INTEREST

None known

SOURCES OF SUPPORT

Internal sources

- University of Teesside, UK.

External sources

- No sources of support supplied

NOTES

Currencies converted on 21st September 2004

Exchange rate: 1 US dollar = 0.821558EUR (\$1 = EURO0.821558)

www.x-rates.com was used to convert

INDEX TERMS

Medical Subject Headings (MeSH)

*Diet; Diabetes Mellitus, Type 2 [*prevention & control]; Energy Intake; Exercise; Randomized Controlled Trials as Topic

MeSH check words

Adult; Humans